HOUR-GLASS STRICTURE OF THE STOMACH AND IRON DEFICIENCY

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" . . . non contenti . . . ulcera agitare, rerum quoque naturam ex aliqua parte scrutati sunt." Celsus. De re medica.

THE object of this note is to put forward the hypothesis that hour-glass stricture of the stomach occurs in persons with a chronic simple gastric ulcer of the lesser curvature, because these persons also have chronic iron deficiency, and that the stricture would not occur without the co-existence of the iron deficiency. Seven cases are described. An association with Plummer-Vinson stricture of the cervical oesophagus and with duodenal stricture is recorded. The X-ray appearances are reproduced. The haemoglobin and serum iron estimations are recorded. Gastroscopy was done in one case. One case was operated on.

Important reviews of hour-glass stomach include those of Moynihan (1904) Hurst and Stewart (1929) and Bockus (1943).

Hour-glass deformity of the stomach occurs almost exclusively in women. It is due to a stricture of the body of the stomach, by which the greater curvature is deeply drawn in medially towards a point on the lesser curvature. At that point there is, or has been, a chronic simple ulcer. It is probable that the adhesion of the gastric ulcer to adjacent structures provides a fixed point to which the contracting scar draws in. The stricture does not occur unless there has been at some time a chronic simple ulcer. It divides the stomach into an upper and a lower loculus, neither of which is contracted or sclerosed. The passage between the two loculi may get very small, but total obstruction, if it ever occurs, must be rare. Hour-glass stomach does not correctly describe the appearance, because the stomach is not waisted symmetrically at the mid-point of its lumen, but rather eccentrically at the lesser curvature. The appearance is reminiscent of that of the Plummer-Vinson stricture in the cervical oesophagus as seen in lateral X-ray views. Normally a chronic simple gastric ulcer heals with a little local scar. The scar that causes the hour-glass narrowing is an abnormal scar, and is submucosal.

The gastric ulcer is not often very active, but it rarely heals, and some ulcer pain is a regular feature. Difficulty in eating is common. This is partly because of the small size of the upper loculus which produces "small stomach" symptoms, and partly because of obstructive feelings. These the patient may interpret as difficulty in swallowing. Impaired nutrition and impaired strength follow. Plummer-Vinson stricture may co-exist, to add cervical oesophageal dysphagia to the difficulty in eating, and to add a particular difficulty in eating meat. As a rule the gastric mucosa is hypotrophic or atrophic. Gastric mucosal secretory activity is low. Malignant change at the site of the scar does not seem to happen. This is an important difference from Plummer-Vinson stricture.

The incidence of hour-glass stomach has fallen of recent years. This may be due to a fall in the incidence of gastric ulcer, to improved treatment of iron deficiency, to better medical treatment of gastric ulcer, or to earlier surgical

treatment. It is uncommon now in this country for a chronic simple gastric ulcer to go for long unhealed and not operated on.

The stricture is usually discovered at a barium meal examination. It is seldom suspected clinically. Examination with a barium meal should include views in the supine and prone positions. If the stricture is extreme, it will be seen in the erect position. If it is not, it may easily be missed, though the associated ulcer may be identified. The supine view is the most valuable. It regularly demonstrates the stricture when it cannot be seen in erect pictures or in prone pictures. This because in the supine position the barium distends the upper end of the lower loculus and the lower end of the upper loculus, and the gap between is easily seen. This is important both for the initial examination, and for subsequent examinations when the ulcer and the stricture are being reviewed. Occasionally in the erect position a bulge of the upper sac may obscure the narrow area. It is less likely that, when the patient is lying down, a bulge of the lower sac may overlie it. Sometimes the delay occasioned by a very narrow passage keeps not only the upper sac but also the oesophagus full of barium for longer than usual. Nevertheless, there is no obstruction at the gastro-oesophageal junction. Hour-glass deformity is sharply distinguished from the other scar complications of gastric ulcer. It is not at all like sclerosis of the lesser curvature or "coffee pot" stomach (which is also a complication of lesser curvature gastric ulcer) and not like the concentric contraction of the antrum, which is a complication of antral ulceration. Neither these nor carcinoma are likely to give any difficulty in diagnosis of the hour-glass stricture. Rarely there may be a considerable deformity of the mid-stomach due to extensive or multiple simple ulcers, but this too is not confusing. The appearance of hour-glass stricture is highly distinctive and is constant. Once the stricture is formed, it never goes away, whether the ulcer heals or not.

Case Reports

Case 1. C.H. Married woman. Born 1931.

She had three children one of whom died of tuberculous meningitis. Her husband is said to have pulmonary tuberculosis. Her first Royal Victoria Hospital attendance was in 1960 when she was twenty-nine. She was complaining of upper abdominal pain and anorexia. Her weight was ninety-one pounds. Because of the family history and of a sedimentation rate of 95 mm. in one hour, attention was directed to tuberculosis as a possible cause of her complaints, but none was found. Her haemoglobin was 71 per cent. Red cells 3.9 millions. M.C.V. 88 cubic microns. M.C.H.C. 30 per cent. She was advised to take iron but her haemoglobin in November 1960 was 67 per cent and in December was 71 per cent. In November 1961 a barium meal (figure 1) showed a chronic simple lesser curve gastric ulcer. In June 1965 she came to medical extern because of "lumps on the legs". Weight was one hundred and sixteen pounds. Sedimentation rate 2 mm. in one hour. Haemoglobin 80 per cent. Serum iron 45 micrograms per 100 ml. Barium meal (figure 2) showed marked hour-glass deformity. She did not consider the menstrual loss remarkable.

Summary: This case shows progression from having the ulcer only to development of the stricture in five years. It is probable that the patient was iron deficient all that time.

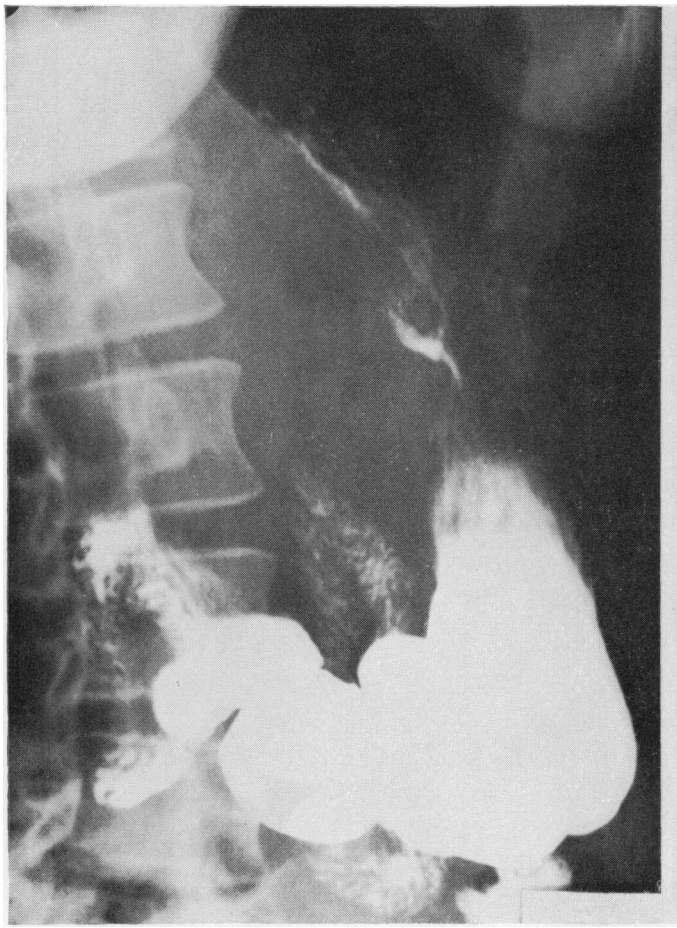

FIG 1. *Case* 1, *in* 1961, *showing lesser curve gastric ulcer*

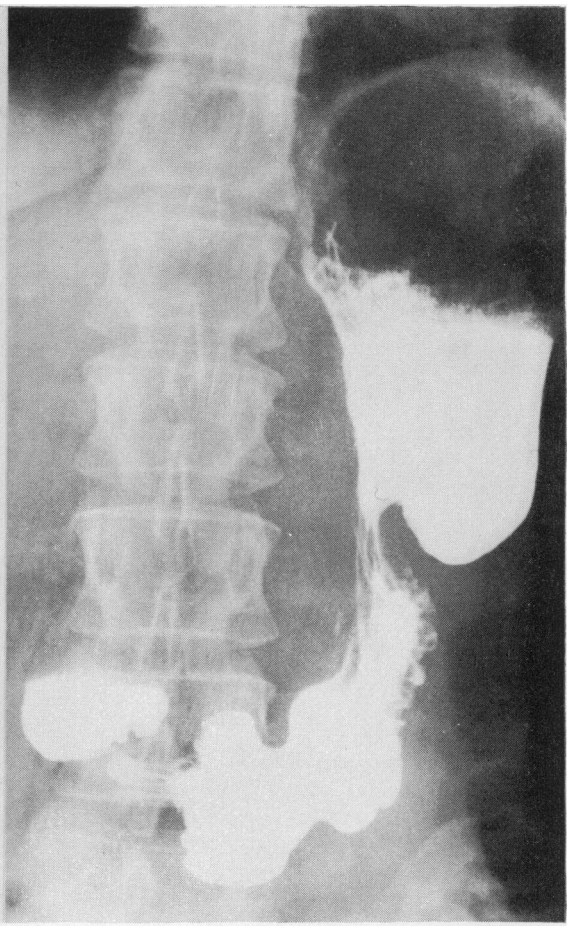

FIG 2. *Case* 1, *in* 1965, *showing hour-glass stricture*

Case 2. E.C. Single woman. Born 1898

She first came to medical extern in 1958 complaining of upper abdominal pain and vomiting. She had had "indigestion" for thirty years, and the pain had lately been bad. There was a doubtful history of rheumatism in the past. Weight was one hundred and seventeen pounds. The barium meal (figure 4) showed a chronic simple lesser curvature gastric ulcer with an hour-glass stricture. The mucosa was hypotrophic. A duodenal web could be seen (figure 3) as a deep narrow incisura of regular outline drawn into the lesser (upper) curvature of the cap from the greater (lower) curvature. In 1960 she came to medical extern complaining of epigastric pain. Weight was one hundred and nineteen pounds. She had been taking an aspirin powder for headache. Haemoglobin 71 per cent. Red cells 4.6 millions per c.mm. M.C.V. 84 cubic microns. M.C.H.C. 28 per cent. The barium meal showed again the ulcer, the adjacent narrowing, and the duodenal web (figure 5). In 1961 she was still having attacks of pain, and the barium meal showed the hour-glass constriction (figure 6). Weight was one hundred and twenty-six pounds. In 1962 the pain was bad. Weight was one hundred and eighteen pounds. In 1963 the pain was bad and she was admitted for surgical treatment. Haemoglobin 80 per cent. Serum iron 99 micrograms per 100 ml. Weight one hundred and seven pounds. No test meal was done. At operation a penetrating

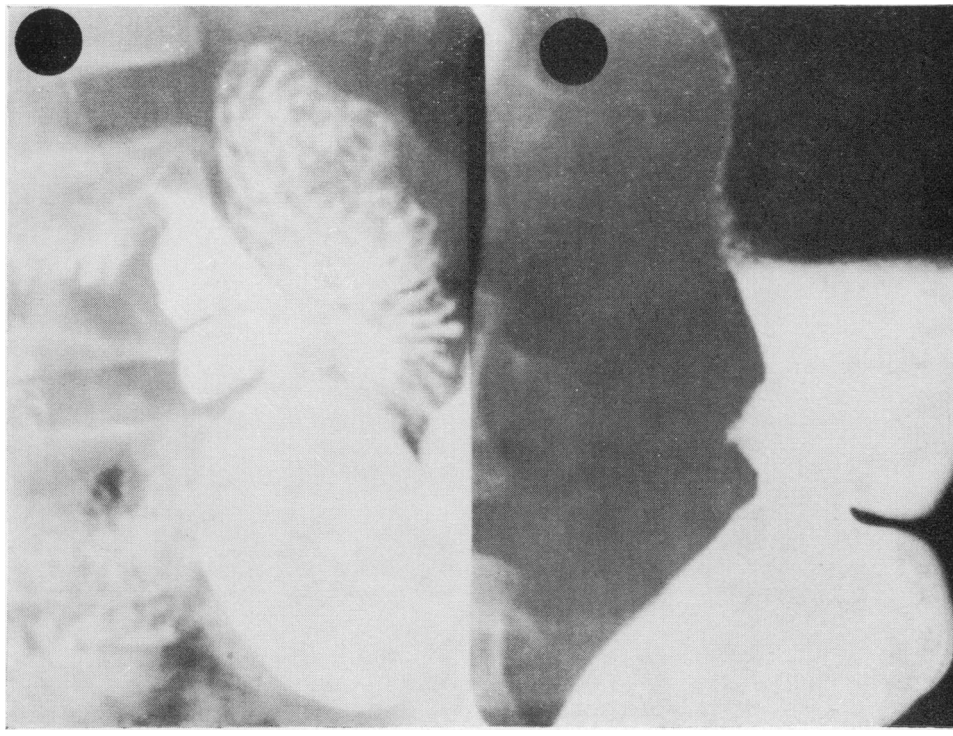

FIG. 3. *Case* 2, *in* 1958, *showing duodenal stricture*

FIG. 4. *Case* 2, *in* 1958, *showing lesser curve gastric ulcer and hour-glass stricture*

FIG. 5. *Case* 2, *in* 1960, *showing the duodenal stricture*

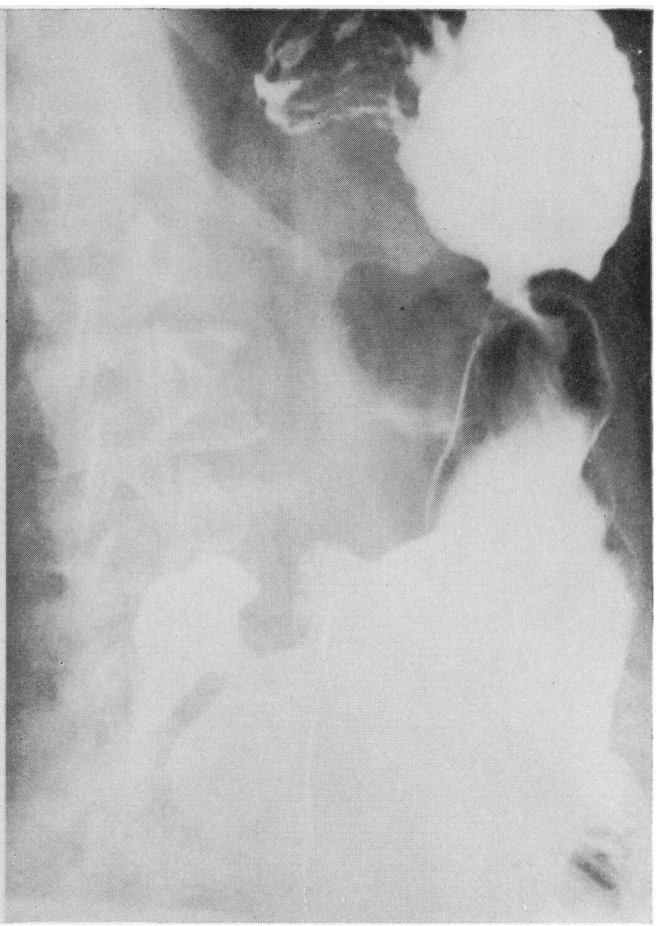

FIG. 6. *Case* 2, *in* 1961, *showing the hour-glass stricture*

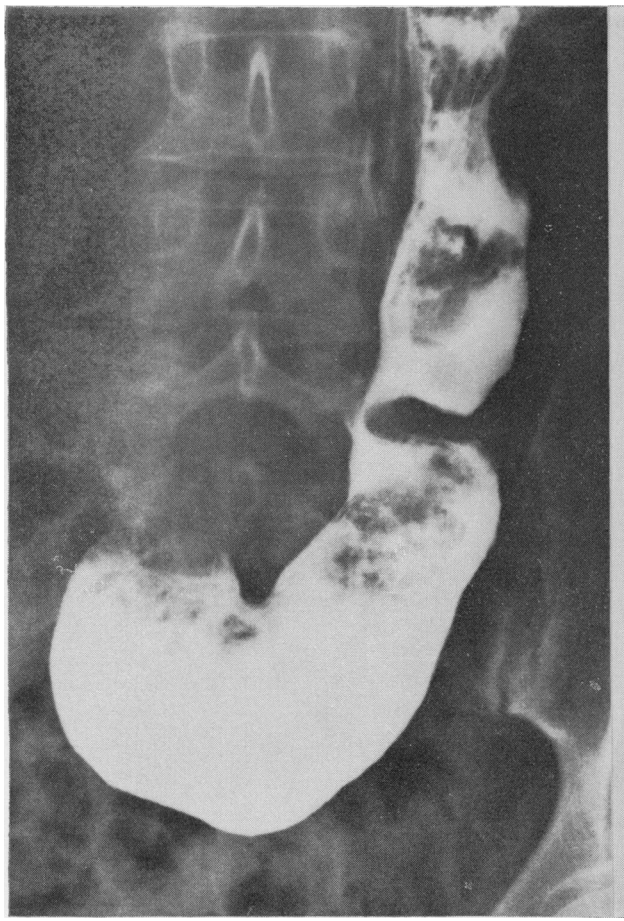

FIG 7. *Case 3, in 1965, showing the hour-glass stricture, erect*

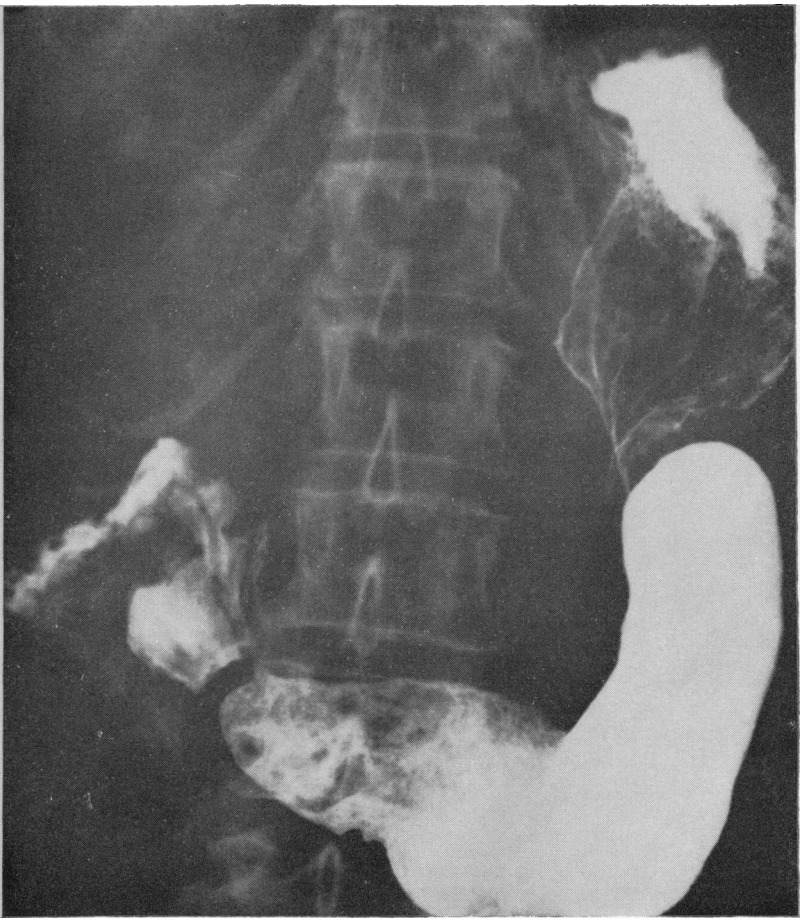

FIG. 8. *Case 3, in 1965, supine view showing the upper end of the lower loculus filled*

gastric ulcer was found with the stricture, corresponding to the X-ray appearances. A partial gastrectomy of Bilroth I type was done by Mr. Morrison, with a gastro-duodenal anastomosis.

Summary: Chronic simple gastric ulcer, hour-glass deformity, stricture or web in the first part of the duodenum, iron deficiency, anaemia and calcified fibroid. The duodenal web was present at the first examination. The hour-glass stricture was also present then.

Case 3. F.McA. Married woman. Born 1896. 2-para.

She first came to the Royal Victoria Hospital in 1964, complaining of loss of weight and abdominal discomfort of thirty years standing. She had no dysphagia. The heart sounds were normal. Haemoglobin 77 per cent. Red cells 4.0 millions per c.mm. M.C.V. 89 cubic microns. M.C.H.C. 31 per cent. Serum iron 40 micrograms per 100 ml. A gruel test meal showed no free acid. Barium meals showed a probable small lesser curvature gastric ulcer with a marked hour-glass deformity (figures 7 and 8). The mucosa was hypotrophic. She was treated with a soft diet of 4,000 calories and with vitamin supplements and with the injections of iron and cyanocobalamin. After she left the ward she did not follow the planned treatment. In March 1965 the haemoglobin was 83 per cent, the serum iron was 57 micrograms per 100 ml., the weight 103 pounds.

Summary: Lesser curvature gastric ulcer, hour-glass deformity, achlorhydria, iron deficiency, anaemia, monoarthritis of shoulder. It cannot be said when the hour-glass stricture formed.

Case 4. E.A. Married woman. Born 1897.

In 1956 a barium meal examination (figures 9 and 10) showed a lesser curvature chronic simple gastric ulcer. No stricture was demonstrated. At that time her weight was one hundred and fifteen pounds, the haemoglobin was 82 per cent and the M.C.H.C. 30 per cent. In 1958 (figure 11) the narrowing could be seen. The gastric mucosa especially in the antrum seemed hypotrophic. In 1962 she came to medical extern complaining of upper abdominal pain and weight loss. Weight was one hundred and ten pounds. Haemoglobin 62 per cent. The barium meal showed the lesser curvature ulcer and an hour-glass deformity (figure 12). There was a duodenal diverticulum. In 1964 she was admitted to the Royal Victoria Hospital. She had been taking aspirin. Haemoglobin 82 per cent. Serum iron 85 micrograms per 100 ml. A gruel test meal showed a very low acid curve—no specimen containing over 10 m.eq./litre of hydrochloric acid. Barium meal showed the stricture as before, and the mucosa even more had an appearance of strophy (figure 13). In 1965 cervical oesophageal narrowing was demonstrated (figure 14). She was treated by prohibiting aspirin and aspirin compounds, by the injections of iron and cyanocobalamin and by maximum feeding. Since then she has been improved. The haemoglobin is 92 per cent. Serum iron 115 micrograms per 100 ml. Heart sounds normal.

Summary: Chronic simple lesser curvature gastric ulcer with hour-glass stricture, cervical oesophageal narrowing, atrophic gastric mucosa, hypochlorhydria, iron deficiency, anaemia, old right mastectomy, emphysema possibly due to flax dust inhalation, periarthritis of the shoulder. It seems that the stricture formed between the age of fifty-nine and sixty-one.

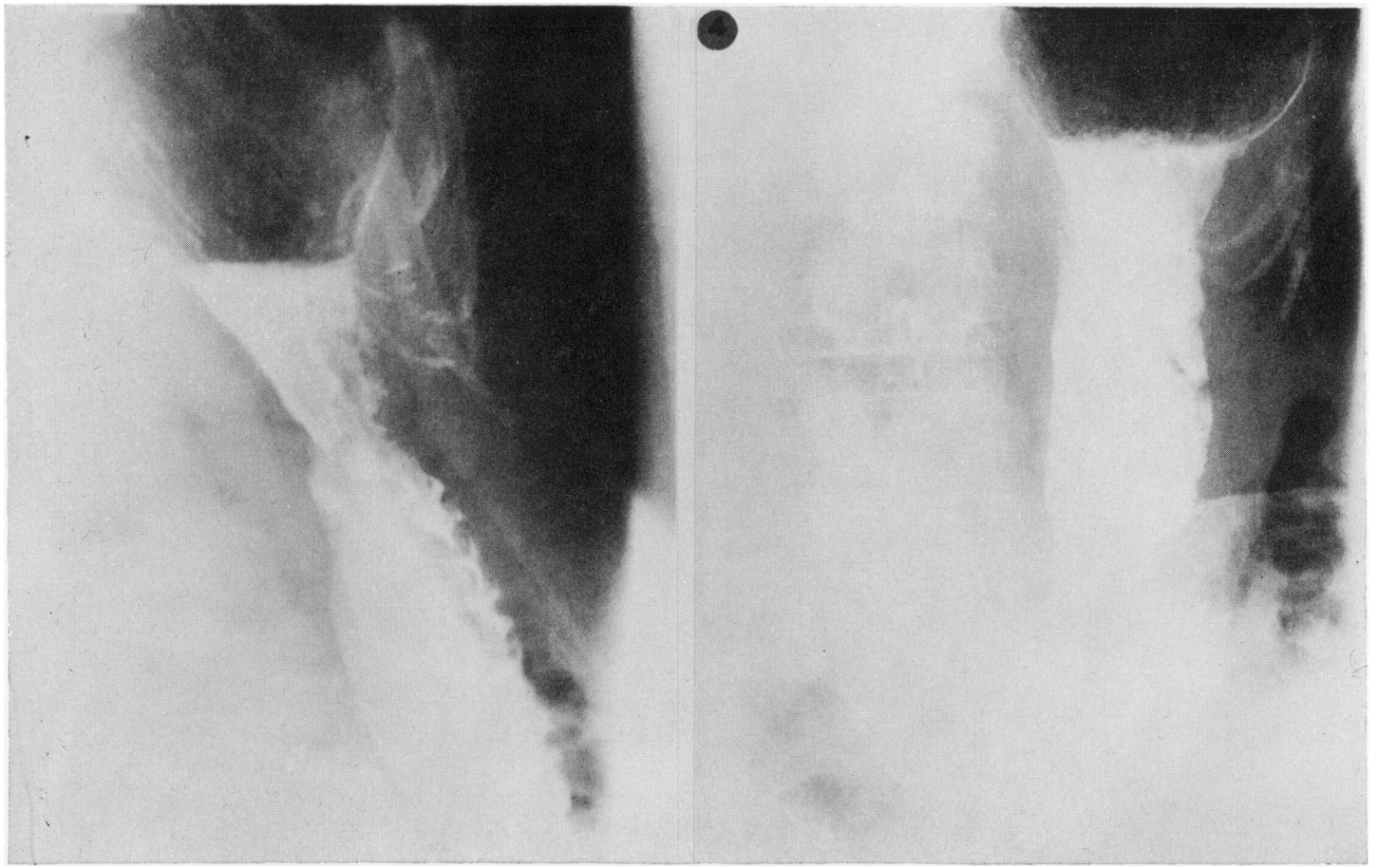

FIG. 9. *Case* 4, *in* 1956, *showing a lesser curve gastric ulcer*

FIG. 10. *Case* 4, *in* 1956, *not showing a stricture*

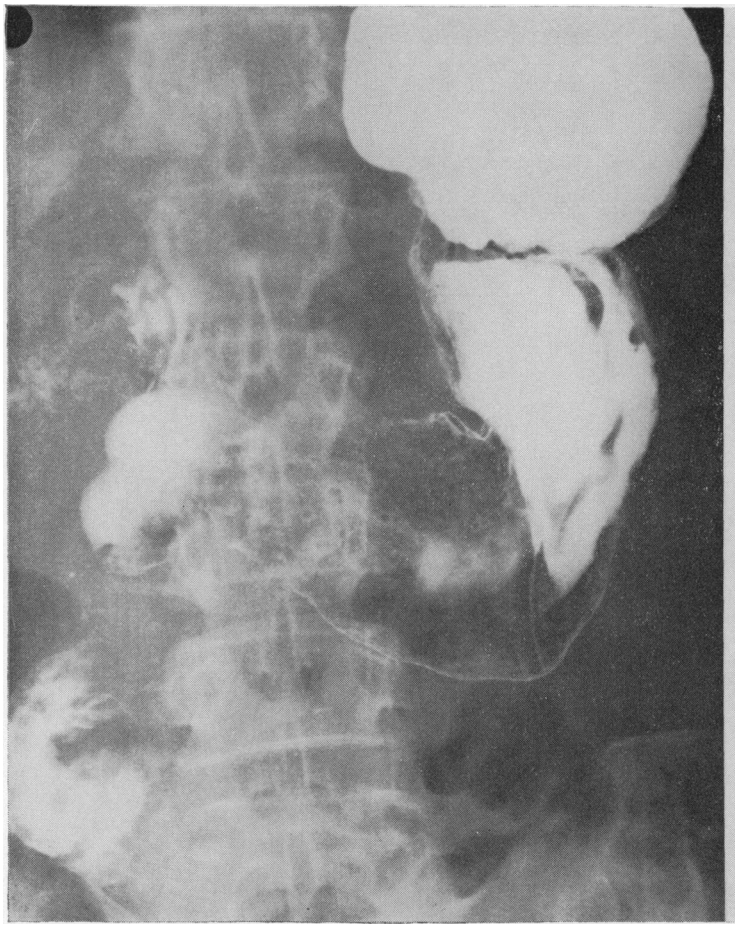

FIG. 11. *Case* 4, *in* 1958, *showing hour-glass stricture.*

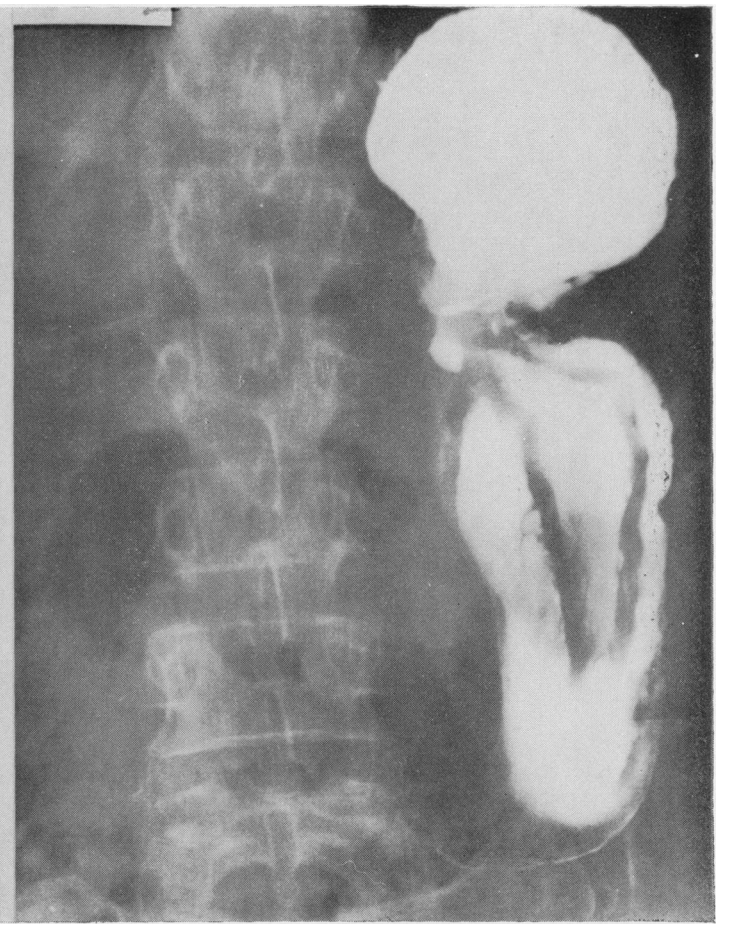

FIG. 12. *Case* 4, *in* 1962, *showing the gastric ulcer and the stricture*

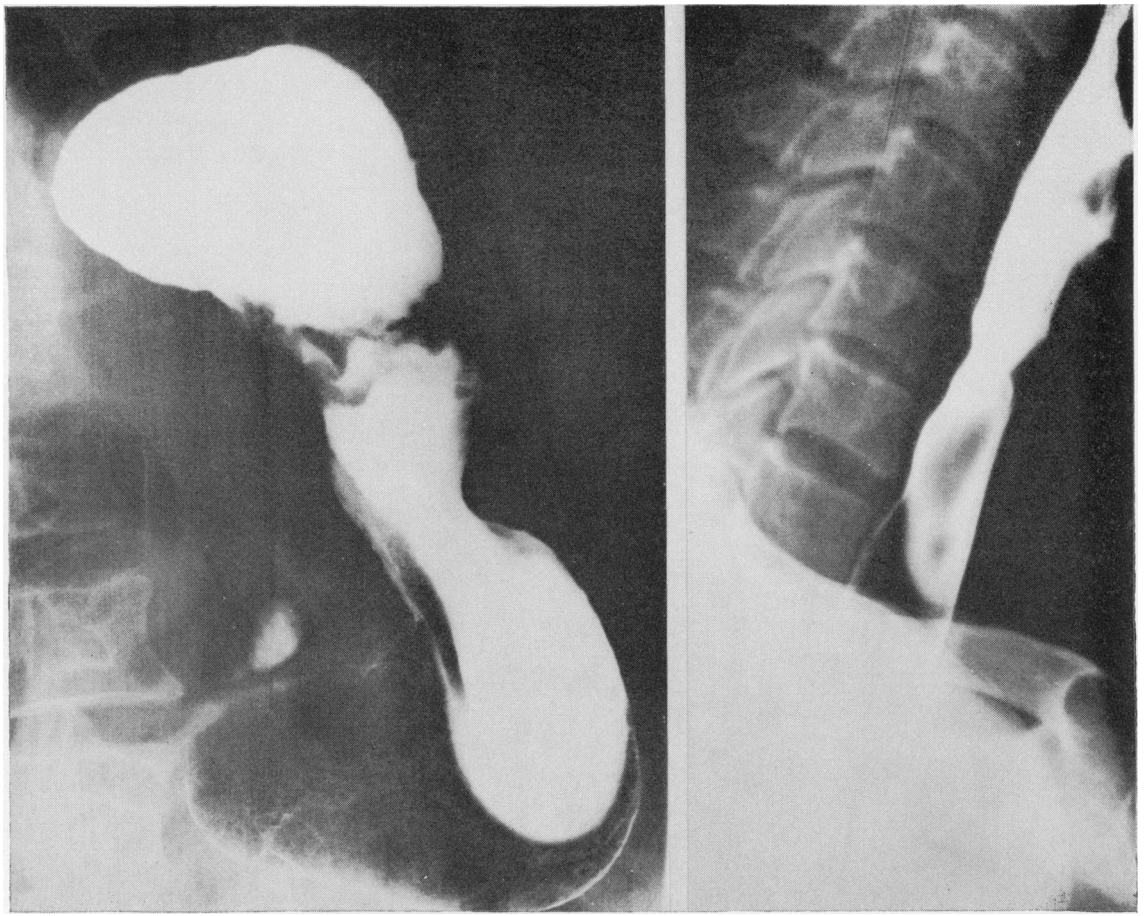

FIG. 13. *Case* 4, *in* 1964, *showing the stricture and hypotrophic mucosa*

FIG. 14. *Case* 4, *in* 1965, *showing cervical oesophageal narrowing*

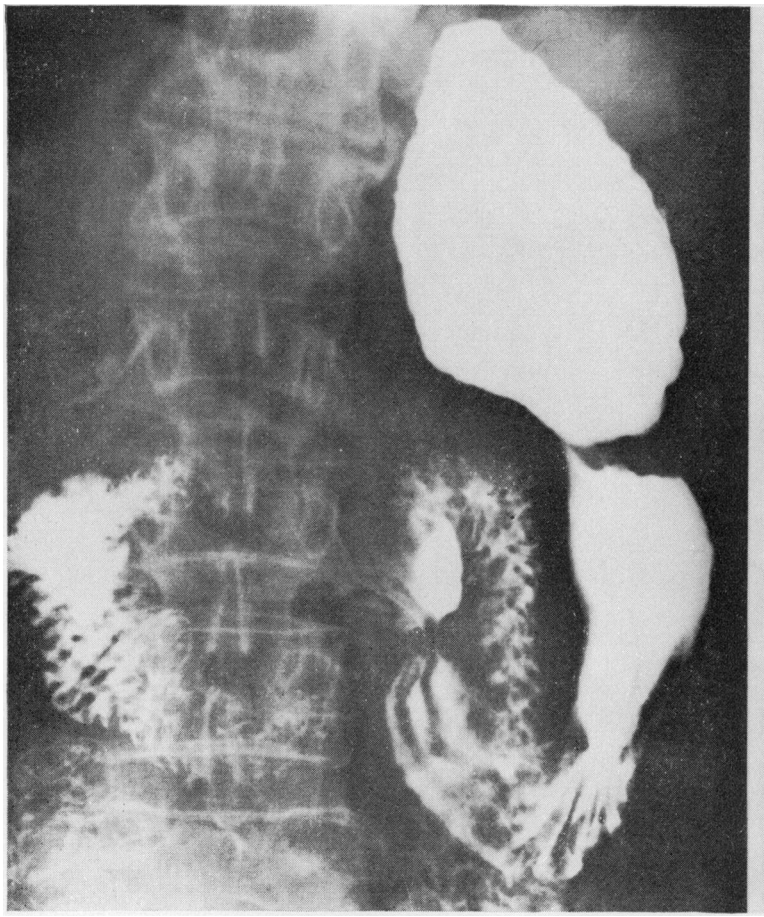

FIG. 15. *Case 5, in 1956, showing the hour-glass stricture*

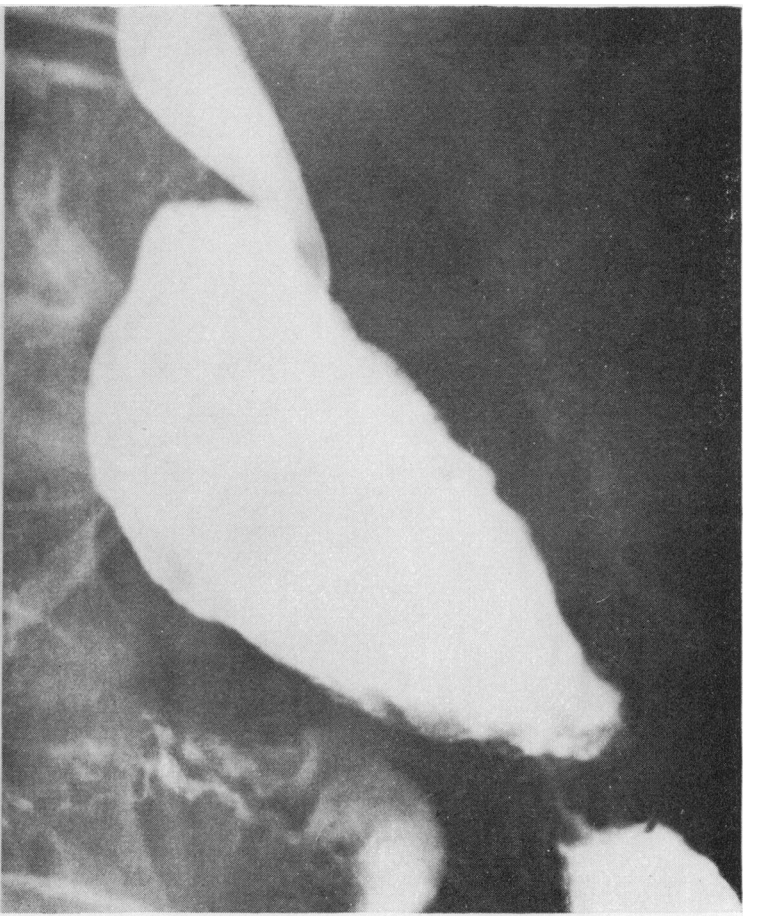

FIG. 16. *Case 5, in 1964, showing hour-glass stricture*

Case 5. M.McC. Married woman. Born 1881. 7-para.

She complained for many years of anorexia, abdominal pain, vomiting after meals, loss of weight and loss of energy. She had had more than one haematemesis in her twenties and thirties. She had been told, after a barium meal examination in 1929, when she was forty-eight, that she had an hour-glass stomach. Her first Royal Victoria Hospital admission was in 1952 when a gruel test meal showed no free acid, a barium meal showed an hour-glass deformity, and gastroscopy (the view was only of the upper loculus) gave a view of the constriction. The mucosa was hypotrophic and no ulcer could be seen. Barium meal X-rays are available for 1956 and 1964 (figures 15 and 16). They show the stricture. An ulcer is not seen. The pyloric channel and cap are normal, as is the antrum. The haemoglobin in 1952 was 88 per cent, 102 per cent in 1954, 105 per cent in 1956, 92 per cent in 1960, and 94 per cent in 1964. On review, in 1964, when she was admitted for congestive heart failure, the serum iron was 40 micrograms per 100 ml. She described some dysphagia which she located in the cervical oesophagus. X-rays showed some narrowing in the cervical oesophagus (figure 17). There was evidence of mitral regurgitation, as there had been for some years.

Summary: Hour-glass deformity of stomach, cervical oesophageal narrowing, iron deficiency, mitral regurgitation. It seems certain that the stricture formed during the child-bearing years.

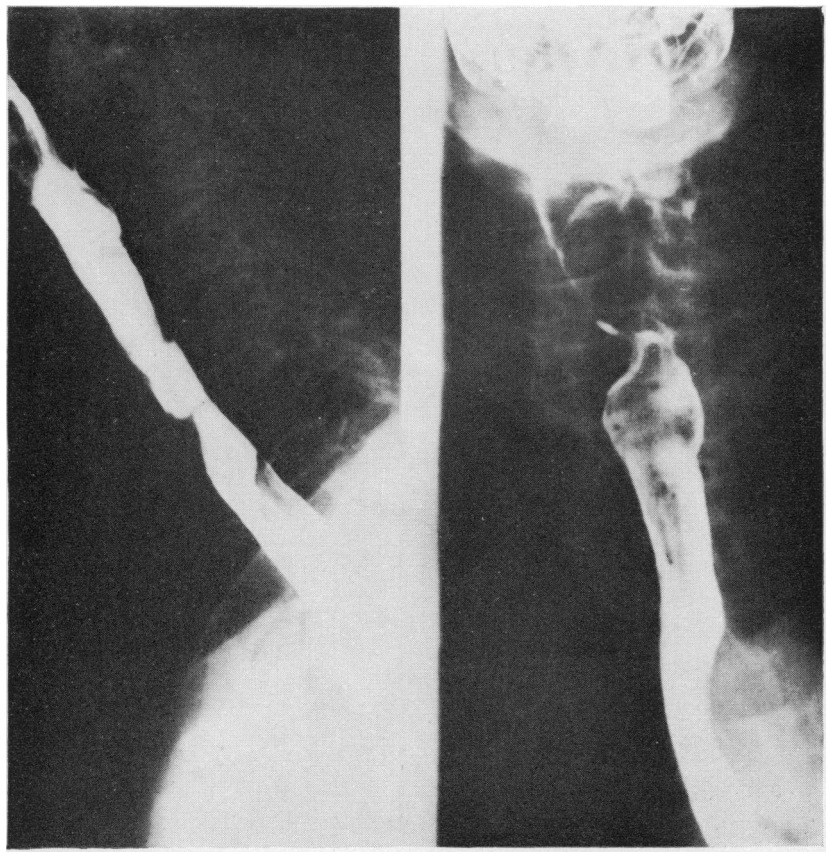

FIG. 17. *Case 5, in 1964, showing cervical oesophageal narrowing*

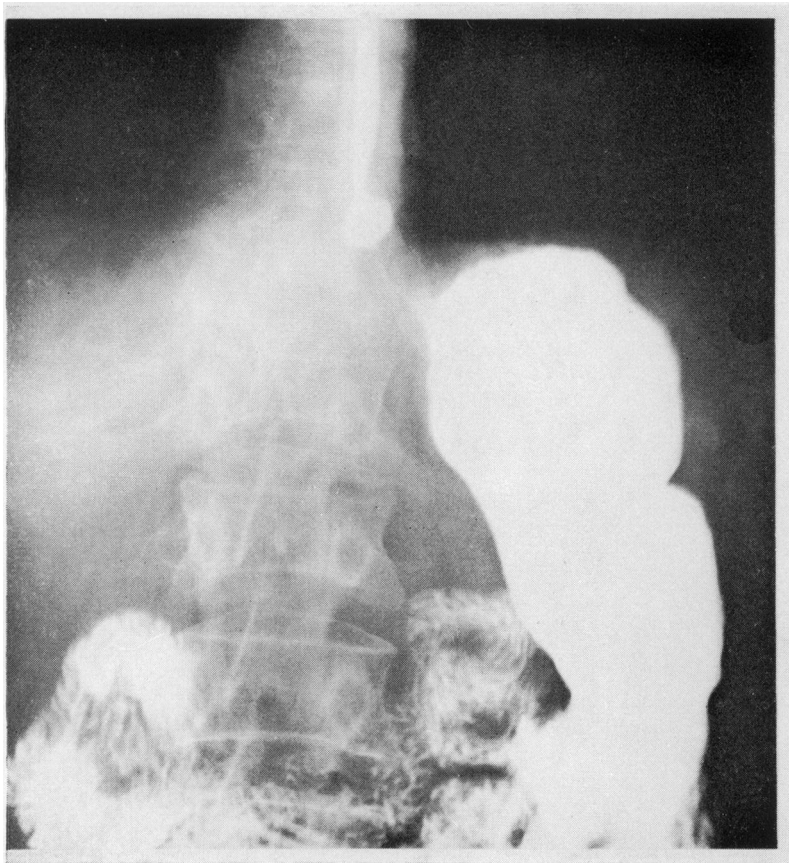

FIG. 18. *Case* 6, *in* 1957, *showing the beginning of the gastric stricture*

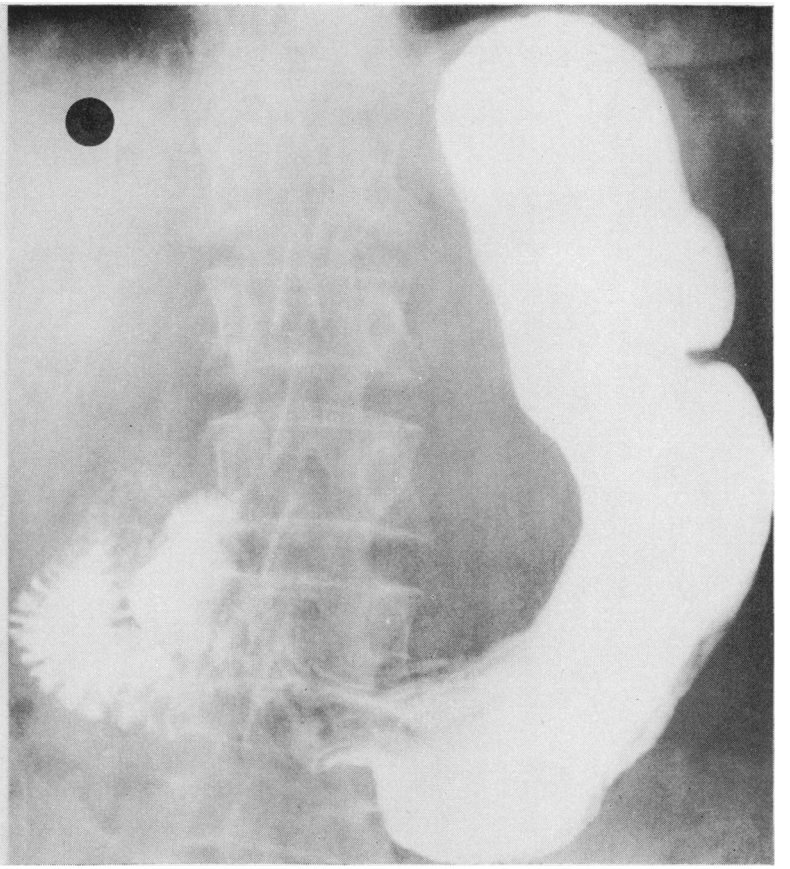

FIG. 19. *Case* 6, *in* 1958, *showing progression of the stricture*

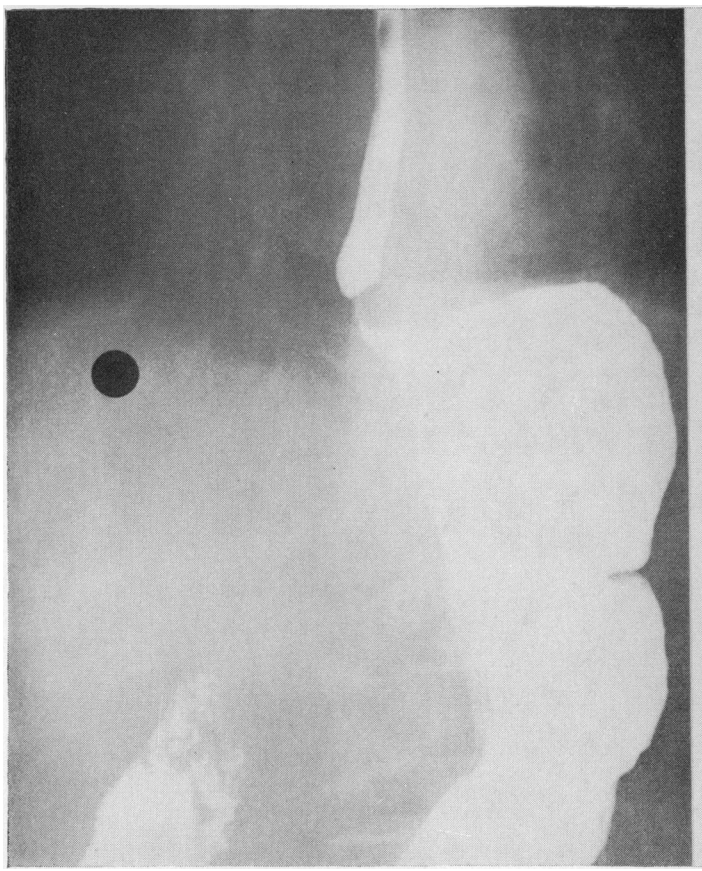

FIG. 20. *Case* 6, *in* 1962, *showing the stricture, hardly more advanced than in* 1958

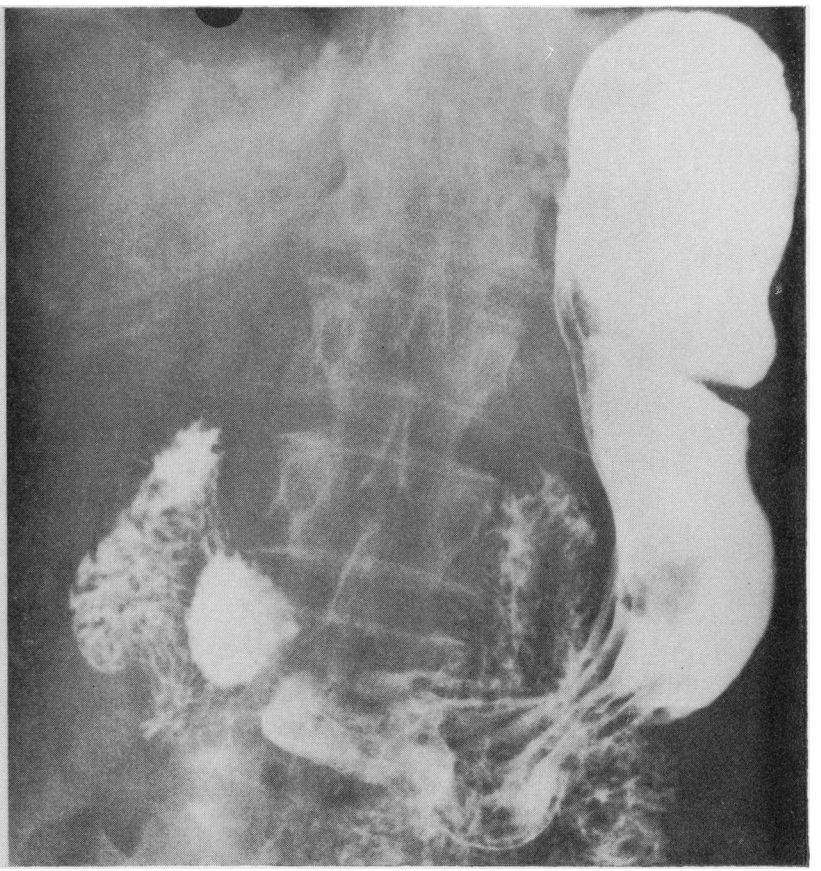

FIG. 21. *Case* 6, *in* 1965, *showing the stricture more advanced*

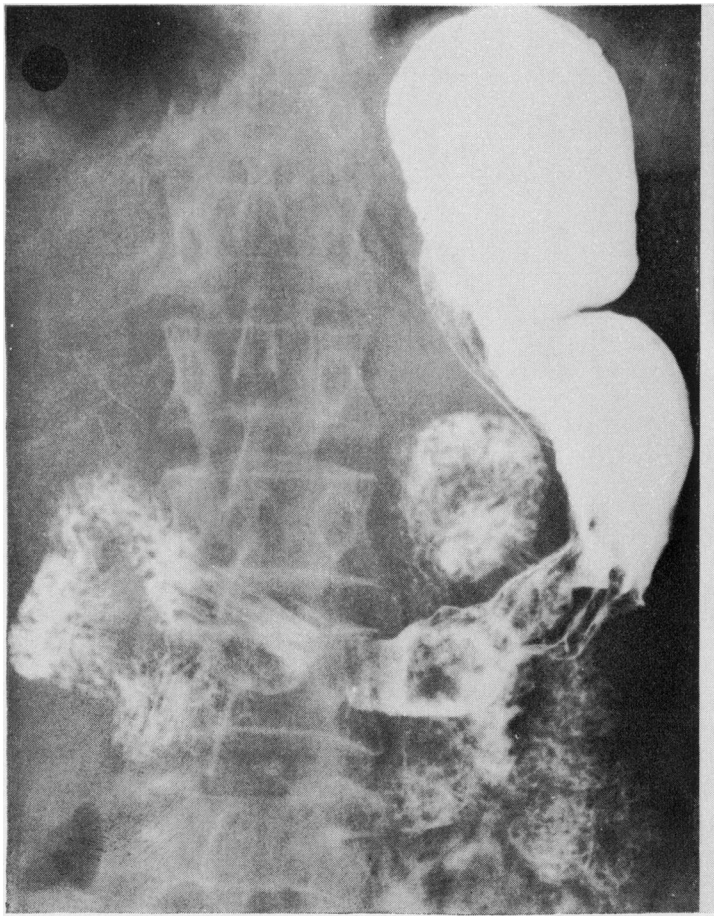

FIG. 22. *Case 6, in 1965, another view of the stricture*

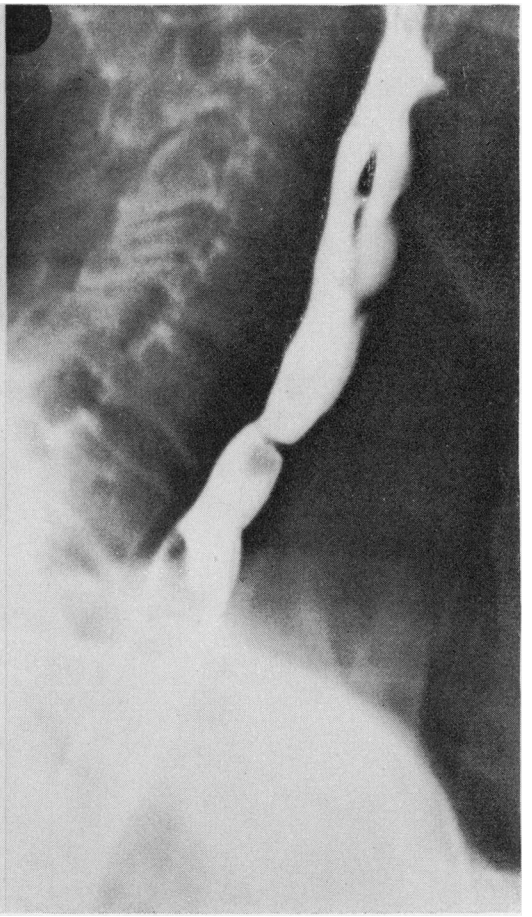

FIG. 23. *Case 6, in 1965, a view of the cervical oesophageal strictures*

Case 6. M.M. Widow. Born 1899. 3-para.

She was first examined in 1949 when she was complaining of a cervical oesophageal dysphagia which was shown to be due to Plummer-Vinson stricturing. She was reported as Case 2 in a previous paper (Bingham and Logan, 1953). Her doctor had treated her for anaemia, sore mouth and angular hacks with iron, including an injection of iron, and vitamins. Her general condition improved but the dysphagia was severe. The Plummer-Vinson stricture was dilated in 1949, and, because the dysphagia recurred, on several occasions since. She was treated off and on with iron, and with cyanocobalamin. In 1957 she mentioned that food "lay" in the upper abdomen, and there was some pain. However, this was transient, and epigastric pain has never been a trouble. In 1959 Mr. Bingham observed at oesophagoscopy that there seemed to be some narrowing in the lower oesophagus. In 1962 it seemed that only a fine bougie could be passed into the stomach. Barium meal examination at those times did not show disease of the lower oesophagus. She was reviewed in 1965 because the cervical oesophageal stricture had recurred. This is seen in figure 23. The stomach at this time was seen to have an indentation of the greater curvature (figures 21 and 22) and on review of the plates it could be seen in those of 1957, 1958 and 1962 (figures 18, 19 and 20). This is believed to be a partial hour-glass stricture. That it did not progress may have been due to partial correction of the iron deficiency, or to a lesser curvature ulcer being transient only. Having the Plummer-Vinson stricture may have protected her from the ulcerogenic effect of aspirin and iron tablets, because she only intermittently could swallow them after a dilatation, and then only for a little while, till the stricture recurred. Her haemoglobin in 1949 was 103 per cent. In 1954 it was 90 per cent. In 1956 it was 84 per cent and the M.C.H.C. was 33 per cent. In 1959 it was 85 per cent. In 1965 it is 82 per cent, the M.C.H.C. 30 per cent and the serum iron (the first ever done) 50 micrograms per 100 ml. It is possible that her iron deficiency was never corrected.

Summary: Plummer-Vinson stricture of the cervical oesophagus, partial gastric stricture, iron deficiency.

Case 7. J.K. Single. Age 92.

This patient was admitted because of a recurrence of abdominal pain. She was transiently jaundiced. Probably the illness was due to gallstones. She had had several hospital admissions and attendances, partly because of her age and infirmity, and partly, among other things, for abdominal pain. The first barium examinations include only erect pictures. The presence of a gastric abnormality may be suspected in the plate of January 1958 (figure 24). A lesser curvature gastric ulcer is seen in April 1958 (figure 25). A supine picture in August 1959 (figure 26) shows the hour-glass stricture well. In 1965 (figure 27) the stricture is still seen. In January 1958 a gruel test meal showed no more than 10 milliequivalents of free acid in any specimen. The haemoglobin, in 1958, was 91 per cent and the M.C.H.C. 31 per cent. In July 1959 the haemoglobin was 81 per cent and the M.C.H.C. 30 per cent. In August 1959 the haemoglobin was 85 per cent and the M.C.H.C. 31 per cent. In November 1959 the haemoglobin was 86 per cent, the M.C.H.C. 32 per cent. In November 1965 the haemoglobin was 84 per cent, the M.C.H.C. 32 per cent and the serum iron was 51 micrograms per 100 ml. In

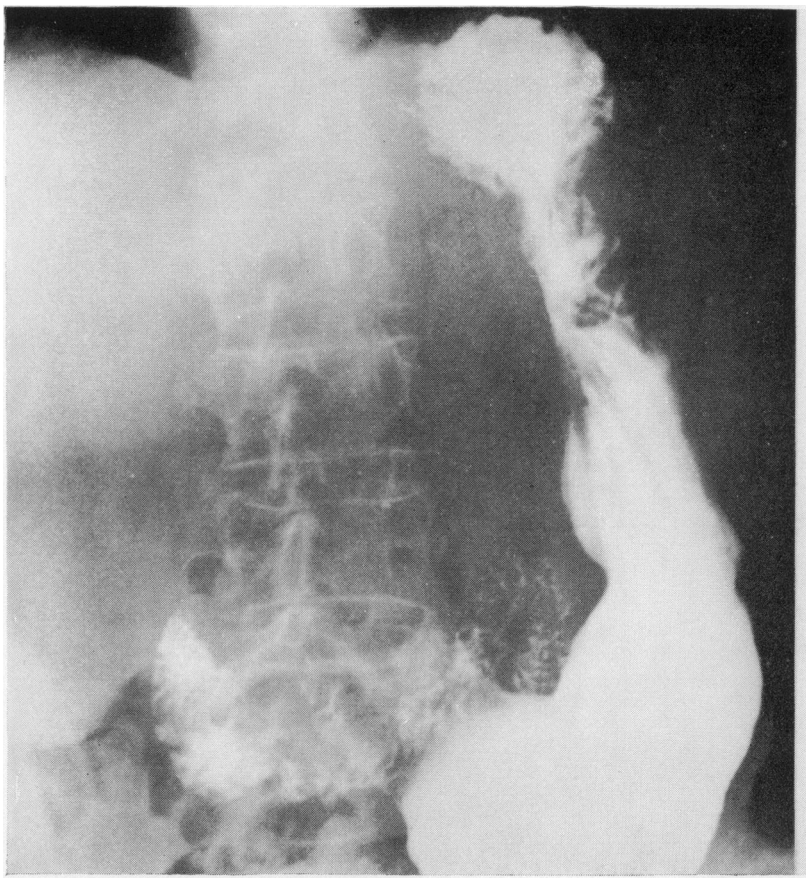

FIG. 24. *Case 7, in 1958, erect view, suspicious of gastric abnormality*

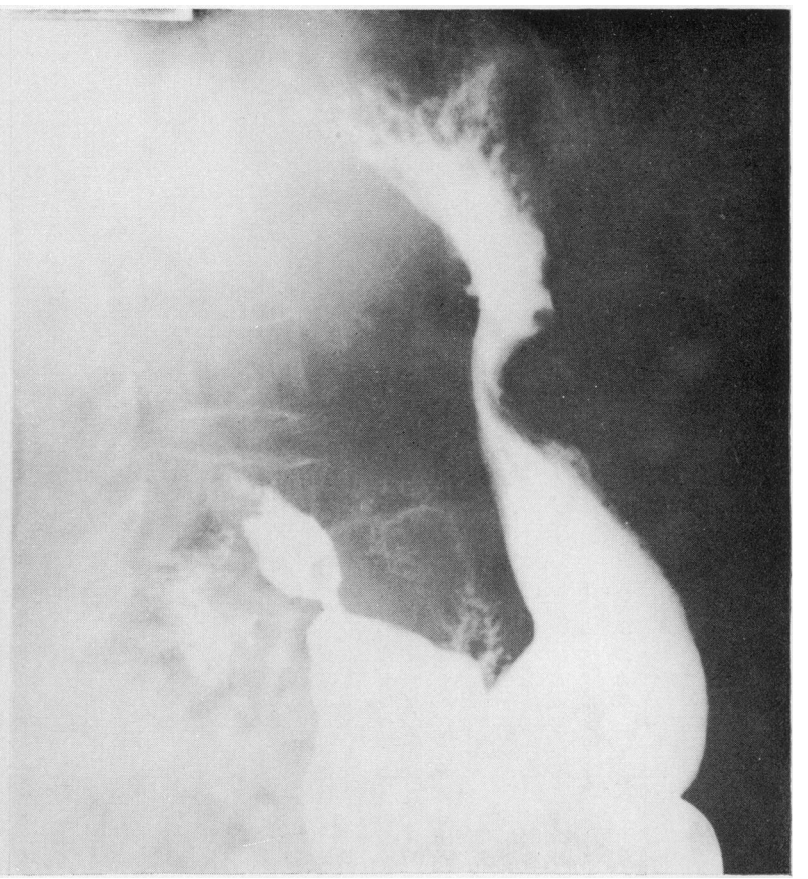

FIG. 25. *Case 7, in 1958, showing a lesser curve gastric ulcer*

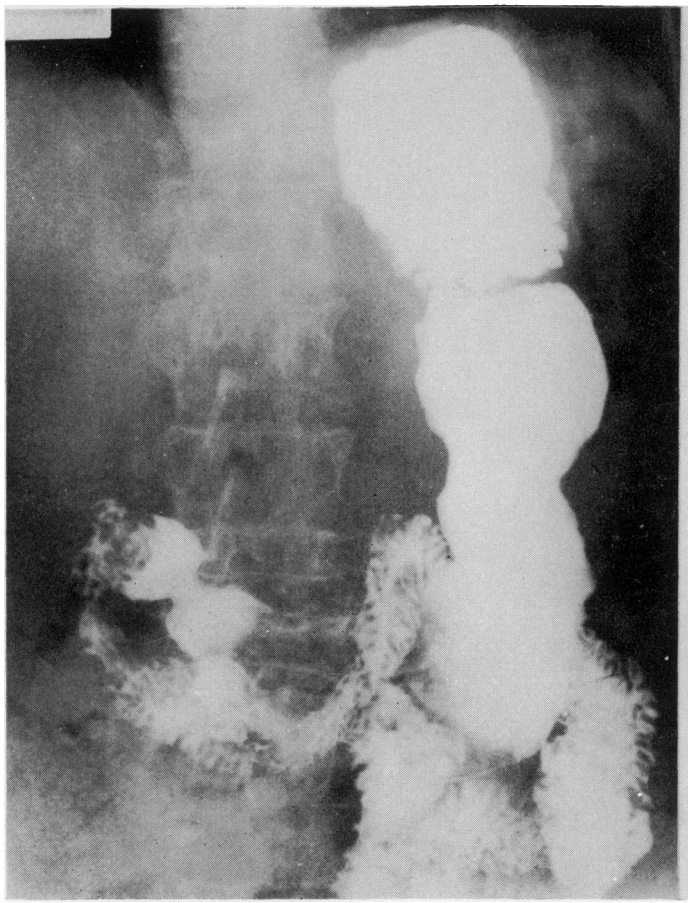

FIG. 26. *Case 7, in 1959, supine view showing an hour-glass stricture*

FIG. 27. *Case 7, in 1965, the stricture is still seen*

July 1959 she had cyanocobalamin injections and in December 1962 she had injections of Imferon. It cannot be said when the hour-glass stricture formed.

Summary: Lesser curvature gastric ulcer, hour-glass stricture, iron deficiency.

Discussion

In view of the association described between hour-glass stricture of the stomach and Plummer-Vinson stricture of the oesophagus and stricture of the duodenum, it is reasonable to think that some general tendency to stricturing in the upper alimentary tract exists in these patients, and that the general tendency occasions abnormal submucosal scarring when, and only when, some local lesion appears, such as the lesser curve ulcer in the case of the stomach. One may think some similar if inconspicuous local lesion occasions the similar scarring in the cervical oesophagus and in the duodenum. All three sites are probably points of special impact of food and drugs, or in the case of the duodenum of the gastric expulsive stream. The question is, is it a reasonable hypothesis that iron deficiency is the cause of the general tendency to stricturing? Is it responsible by itself (or inter alia) for the development of the hour-glass scar in certain cases of lesser curvature gastric ulcer? The hypothesis finds support in the evidence of iron deficiency in the cases reported, in the co-existence of the Plummer-Vinson stricture long thought to be caused by iron deficiency, in the exclusive or almost exclusive incidence in women, and in a clinical impression that iron administration facilitates the normal healing of lesser curvature gastric ulcers. It would be helpful if in future all case records of hour-glass stricture detailed the history of the iron state. Reports of cases of lesser curvature gastric ulcer in patients with long continuing iron deficiency which did *not* form an hour-glass stricture would also be helpful in testing the hypothesis.

The duodenal stricture in case 2 is quite unlike chronic duodenal ulcer deformity, and does resemble hour-glass stricture of the stomach and the Plummer-Vinson stricture. Moyhihan's case XV had what he described as an hour-glass duodenum as well as two gastric strictures. The form of duodenal stricture however is not clear from his report. It could have been as in the present case, or it could have been a duodenal ulcer wasp-waist deformity (Gutmann 1947).

The almost exclusive incidence in women is of great importance, though not conclusive. It cannot be claimed that an incidence predominantly female is necessarily mediated by menorrhagic iron deficiency. This is, however, one of the agents important in making a disease predominantly female in incidence, and so far no other is recognisable in these cases.

It might be said that the iron deficiency is at least in part in many cases due to aspirin taking. This may be so. Such iron deficiency would have the same effect as that due to uterine haemorrhage. No doubt aspirin taking accounts for some cases of postmenopausal iron deficiency. It is probable too that aspirin as a tranquilliser and hypnotic, and perhaps also as an analgesic, is taken more by women than men. Still many men regularly take aspirin, and yet hour-glass stricture is extremely rare in men. Aspirin may cause gastric ulceration, acute and chronic, in both sexes, but again the stricture is rarely found except in women. Some factor above and beyond having an ulcer and taking aspirin must be responsible. It is not a high secretion of acid and enzymes by the gastric mucosa, because the mucosa in these cases is hypotrophic or atrophic. Indeed a gruel test meal shows

that some are hypochlorhydric or achlorhydric (cases 3, 4, 5 and 7). One cannot say whether the hypotrophic mucosa is a causative factor in the forming of the stricture, nor whether the hypotrophy could be caused by iron deficiency. These reports provide no information in that respect.

It might be said that the bleeding and anaemia and iron deficiency are only to be expected of a chronic gastric ulcer, and that the association of gastric ulcer and anaemia is therefore not remarkable, and the anaemia not necessarily due to uncompensated uterine loss of haemoglobin and iron. It would still be possible that the iron deficiency so produced made for chronicity of the ulcer, and also for the formation of the stricture. It is probable, however, that the incidence of bleeding in chronic gastric (and in chronic duodenal) ulcer has been over-estimated. It is now common ground that many of the cases of haematemesis and melaena admitted to medical wards have been occasioned by aspirin erosions of the gastric mucosa, *and that this may be so even if the patient has a co-existing chronic simple gastric (or duodenal) ulcer*. When chronic gastric ulcers bleed, they bleed severely but it is not often.

One must draw attention to the difficulty in studying the effects of iron deficiency on the formation of cervical oesophageal stricture and gastric stricture. These cases commonly come to notice when the stricture is well established, and sometimes years after it is well established. Six of the seven cases described were post-menopausal, though in case 4 the stricture seems to have formed after the meno-pause. Once the menopause occurs, the anaemia may in part or in whole correct. The serum iron may partly or wholly return to normal. Much depends on the aspirin-taking habits of the patient, and much on what treatment with iron she received. In examining postmenopausal patients it is hard to get satisfactory information about the iron state at the time the stricture was being formed. Once the stricture is formed, it never goes away, no matter how much the anaemia and iron deficiency are corrected. But in premenopausal patients adequately treated in the end, and in postmenopausal patients, there may be no, or only imperfect, evidence of the iron deficiency which caused the stricture.

When the stricture can be shown to have formed *after* the menopause, there can be no easy presumption that the patient was iron-deficient at the material time, though that may have been the case. Iron deficiency is not rare in postmenopausal women, sometimes because a premenopausal deficiency was never corrected, and somtimes because it was acquired after the menopause, e.g., by aspirin taking. Sometimes it cannot be explained. Nevertheless, the iron deficiency must be demonstrated, and there must be at least a reasonable presumption that it was present at the time the stricture was forming. In case 4 the stricture may have formed after the menopause but there is evidence of iron deficiency. In case 7 the stricture may have formed in old age or may not, but there is evidence of iron deficiency. It is clear enough that in cases 1 and 5 the stricture formed during the childbearing years. In cases 2, 3, 6 and 7 the stricture cannot be dated. The difficulty in dating the formation of the stricture lies partly in a lack of early barium examinations and partly in a lack of supine views.

Estimation of the serum iron is essential, because the serum iron may be low, even when the haemoglobin is within more or less normal limits. It is iron deficiency we are studying and not haemoglobin deficiency. If the mucosal atrophy

progresses, cyanocobalamin deficiency may be added, and this may give rise to a supposition that the stricture is associated with pernicious anaemia.

Fortunately in planning the treatment of chronic simple gastric ulcer it is not nesessary to decide the question of the relationship of iron deficiency to ulcer healing and stricture formation. General principles require that in gastric ulcer, as in most diseases, if iron deficiency exists, it should be corrected—promptly and completely. Estimation of serum iron as well as of the haemoglobin is essential in assessment of progress. The use of an intramuscular injection of iron is necessary at least in the beginning. Both the haemoglobin and the serum iron should be restored to normal, and kept there. Iron tablets lie under suspicion of causing gastric mucosal erosions as aspirins do, and they should not be used in disease of the gastric mucosa. After the injection of iron, treatment may be carried on by a bland liquid preparation of iron, but if there is difficulty one should return to the injection. Aspirin in all its forms and all salicylate preparations should be forbidden because of their corrosive action on the gastric mucous membrane, and their production of haemorrhage and iron deficiency. The treatment of chronic simple lesser curvature gastric ulcer and the prevention of its complications now seem to lie mainly in these two measures, and in maximal and optimal feeding (assuming always that there is no obstructive delay in gastric emptying also to be coped with).

It remains to be seen if this hypothesis of abnormal gastric scarring being due to iron deficiency has any general application to other visceral injuries which heal by scarring. For instance, observation of the serum iron and the haemoglobin in rheumatism ought to show whether rheumatic subendocardial scarring is worse in iron deficient individuals.

Summary

Seven cases of hour-glass deformity of the stomach are described in whom there was also evidence of iron deficiency. One case had also a duodenal stricture and three had strictures of the cervical oesophagus. It is suggested that in such cases a general tendency to stricturing exists, which acts locally to produce a stricture by an abnormal submucosal scar when, for whatever reason, a local ulcer is formed. It is suggested that iron deficiency may be responsible for the stricturing tendency, hindering normal healing of chronic simple lesser curvature gastric ulcer and inducing the formation of the gastric stricture. It is advocated that the treatment of chronic simple gastric ulcer of the lesser curvature (without any delay in gastric emptying) should rest mainly on correcting iron deficiency, on prohibiting aspirin and salicylates, and on feeding which, if bland, is both maximal in calories and optimal in composition. Where the mucosa is atrophic and the diet inadequate, the injection of cyanocobalamin should be added.

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BOOK REVIEWS

URINARY DIVERSION. By Theunis Coetzeen, Ch.M., F.R.C.S., F.C.S.(S.A.), F.A.C.S. (Pp. 36; figs. 48. 10s.). Edinburgh and London: E. & S. Livingstone, 1966.

OPERATIONS for urinary diversion in disease of the urinary bladder are becoming more common and the place for these operations and the possibility they give for a more normal life should be better known. This prize essay by a South African consultant should interest may who are not specialists in this field.

HISTORY OF THE TRUSTEES OF THE HUNTERIAN COLLECTION. By Sir Victor Negus, Hon. D.Sc.(Manch.), M.S.(Lond.), F.R.C.S.(Eng.). (Pp. viii +132. 30s.). Edinburgh and London: E. & S. Livingstone, 1965.

THIS beautifully produced and relatively inexpensive monograph may be of limited interest, but many may read it with greater attention than its title would suggest. Admittedly much of it is concerned with lists of trustees, but it traces the long history of a great institution. It well illustrates the difficulty of running such an institution with a committee chosen more for distinction in other fields of activity and on a basis of age and seniority than for proven interest.

PHOTOELECTRIC COLORIMETRY IN CLINICAL BIOCHEMISTRY. By G. E. Delory. (Pp. 79. 25s.). London: Hilger & Watts Ltd., 1966.

THIS latest book by Dr. Delory is substantially a revision of his previous book "Photoelectric Methods in Clinical Biochemestry", published in 1949. The section on elementary principles and instrumentation has been shortened by excluding both the description of various types of absorptiometers and the transmission values for standard light filters. The latter omission is unfortunate, as a laboratory text book containing the transmission properties of commercially available filters would be most welcome. In the second part of the book, more recent techniques for estimating important biochemical compounds have been described and a list of references for each method is given in the bibliography.

Although the whole subject has been fully treated in modern text books of practical clinical biochemistry, this small book may be useful to students interested in photoelectric colorimetry. S.G.W.